AD-A124 936 CHRONIC CONSUMPTION OF A LOW SODIUM DIET: HORMONAL AND PHYSIOLOGICAL EFFE..(U) ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MA 1/1

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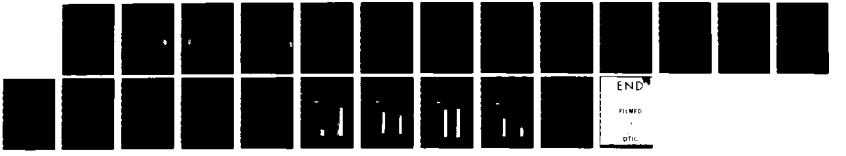

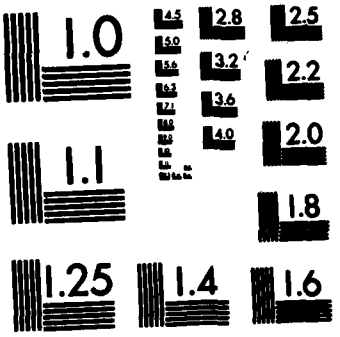

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1. REPORT NUMBER: M7/83	2. GOVT ACCESSION NO.: AD-A124936
3. RECIPIENT'S CATALOG NUMBER:	
4. TITLE (and Subtitle): Chronic Consumption of a Low Sodium Diet: Hormonal and Physiological Effects During Exercise in the Heat	5. TYPE OF REPORT & PERIOD COVERED:
	6. PERFORMING ORG. REPORT NUMBER:
7. AUTHOR(s): R. P. Francesconi, R. W. Hubbard, M. Mager	8. CONTRACT OR GRANT NUMBER(s):
9. PERFORMING ORGANIZATION NAME AND ADDRESS: US Army Research Institute of Environmental Medicine, Natick, MA 01760	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: 3M161102BS10 34382101005 BS10C Sys Hlth Haz Rsch
11. CONTROLLING OFFICE NAME AND ADDRESS: US Army Medical Research & Development Command, Ft. Detrick, MD 21701	12. REPORT DATE: 26 Jan.1983
	13. NUMBER OF PAGES: 13
14. MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office): Same	15. SECURITY CLASS. (of this report): UNCLASSIFIED
	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE:

16. DISTRIBUTION STATEMENT (of this Report)

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17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

Same

18. SUPPLEMENTARY NOTES

NA

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Hyperthermic exhaustion, adrenocortical activity, heat injury

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

To elucidate the effects of sodium deficiency on the ability to work in the heat, immature rats were fed a diet deficient in sodium (Na^+) for approximately 2 months. Rates of weight gain were severely affected ($p<.01$) in the Na^+ deficient rats (1.7 g/day vs. 7.2 g in controls) although fluid consumption was unaffected. The low Na^+ diet effected no alterations in endurance or weight loss during exercise in the heat to hyperthermic exhaustion, but final core and skin temperatures were significantly reduced in the low Na^+ group ($p<.02$) and hemotocrit ratios were significantly ($p<.001$) increased. Circula-

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ting Na^+ and potassium (K^+) levels were significantly ($p<.05$) increased in both groups after hyperthermic exhaustion. In the Na^+ deficient groups, plasma levels of both aldosterone and cortisol/corticosterone were significantly ($p<.05$) increased, and these increments were exacerbated following exercise to hyperthermic exhaustion. Consumption of the low Na^+ diet elicited significant increments in circulating levels of lactate ($p<.01$) and creatinine ($p<.01$), both of which were increased further after exercise. Circulating glucose was unaffected by consumption of the sodium deficient diet but declined in this group following exercise. Urea nitrogen and lactic acid dehydrogenase were increased after exercise but unaffected by the low sodium diet. Additional experiments are planned to elucidate further the metabolic and physiologic effects of chronic ingestion of a low Na^+ diet.

Chronic consumption of a low sodium diet: hormonal and physiological effects during exercise in the heat.

R.P. Francesconi, R.W. Hubbard, and M. Mager
US Army Research Institute of Environmental Medicine
Natick , Massachusetts 01760

Send proofs to:

Dr. Ralph Francesconi
Heat Research Division
USARIEM
Natick, MA 01760

Abstract

To elucidate the effects of sodium deficiency on the ability to work in the heat, immature rats were fed a diet deficient in sodium (Na^+) for approximately 2 months. Rates of weight gain were severely affected ($p < .01$) in the Na^+ deficient rats (1.7 g/day vs. 7.2 g in controls) although fluid consumption was unaffected. The low Na^+ diet effected no alterations in endurance or weight loss during exercise in the heat to hyperthermic exhaustion, but final core and skin temperatures were significantly reduced in the low Na^+ group ($p < .02$) and hemotocrit ratios were significantly ($p < .001$) increased. Circulating Na^+ and potassium (K^+) levels were significantly ($p < .05$) increased in both groups after hyperthermic exhaustion. In the Na^+ deficient groups, plasma levels of both aldosterone and cortisol/corticosterone were significantly ($p < .05$) increased, and these increments were exacerbated following exercise to hyperthermic exhaustion. Consumption of the low Na^+ diet elicited significant increments in circulating levels of lactate ($p < .01$) and creatinine ($p < .01$), both of which were increased further after exercise. Circulating glucose was unaffected by consumption of the sodium deficient diet but declined in this group following exercise. Urea nitrogen and lactic acid dehydrogenase were increased after exercise but unaffected by the low sodium diet. Additional experiments are planned to elucidate further the metabolic and physiologic effects of chronic ingestion of a low Na^+ diet.

Key Words: hyperthermic exhaustion, adrenocortical activity, heat injury.

Introduction

The role of electrolytes in the etiology of heat-induced injuries has been investigated extensively. Knochel (18) summarized data indicating that 46% of 121 heat stroke cases had presented with potassium levels less than 3.5 mEq/L; he concluded from these data that hypokalemia might play a decisive role in the development of serious heat injury. However, Shibolet et al. (26) reasoned that since heat stroke occurs so frequently during the first few days of exercise in a hot environment, before excessive potassium loss is incurred via sweat and urine, it is unlikely that potassium deficiency is an important etiologic factor. However, in a more recent publication Hubbard et al. (16) have demonstrated conclusively in rats that feeding a diet deficient in potassium can severely impair both physical performance and thermoregulatory efficiency during exercise in the heat.

While it is generally accepted that oral administration of a dilute saline solution can relieve the symptomatology of heat-induced muscle cramps, the occurrence of this disorder is evidently rare (26). In many retrospective studies of more severe heat injuries in humans, circulating levels of sodium appear to be within the normal range upon admission (14, 16) or slightly elevated (6, 19, 25) due to dehydration. The process of heat acclimatization in humans is partially manifested in the secretion of a more dilute sweat as a result of sodium retention (27) correlated with increased aldosterone levels and plasma renin activity during recurrent bouts of exercise in a hot environment (7).

The importance of maintaining proper electrolyte levels during exercise in a hot environment has led several investigators to test the value of an electrolyte replacement solution in reducing the physiological cost of work in the heat. Costill et al. (5) concluded that in dehydrated subjects water was as effective as a solution containing sodium, potassium, chloride, and glucose in

maintaining plasma volume and electrolyte balance. In a related study Francis (11) compared the value of no fluid replacement, an electrolyte solution, and water during exercise in the heat. While he demonstrated no advantages of the electrolyte solution over water with respect to plasma volume, hematocrit, heart rate, and several clinical chemical indices of stress, Francis (11) concluded that the electrolyte solution was efficacious in compensating for sodium and potassium losses in sweat and urine. Greenleaf and Brock (13) demonstrated that consumption of a hypertonic saline solution prior to exercise in the heat resulted in a greater plasma volume during exercise in the heat; in these experiments the comparison was made versus an isotonic saline or hypertonic calcium gluconate beverage. However, it should be noted that the greater plasma volume attained after saline drinking did not translate to physiological benefits since rectal temperatures were higher following exercise in the heat after consumption of hypertonic saline.

It is evident that most of the aforementioned reports or studies were performed either retrospectively in heat-injured patients or during acute heat exposure/exercise protocols. No prospective investigations have been reported wherein animals have been fed diets deficient in sodium for prolonged periods and then tested for physical and physiological performance during exercise in the heat. We have used a rat model (15, 17) for human heat stroke to quantitate the effects of several factors which predispose to heat injury (9, 10). In the present study we have examined in this model the effects of prolonged sodium deprivation on the thermoregulatory, physiological, and physical responses to exercise in the heat.

Methods

Immature male rats (139.2 ± 5.7 g, mean ± SD) were purchased from the Charles River Breeding Laboratories (Wilmington, MA). Upon arrival at the laboratory, the rats were placed singly in wire-bottomed cages, and held in a windowless room (fluorescent lighting, 0600-1800h daily) maintained at 22 ± 1°C, 35-45% RH. Within several days these rats were placed on a low-sodium, vitamin-supplemented diet (US Biochem. Corp., Cleveland, Ohio, Catalog Number 21675); they were then allowed ad libitum access to this diet as well as tap water. Quantitative analysis of the test diet revealed an average sodium (Na^+) content of less than 2 mEq/kg; the Na^+ content of the drinking water manifested a Na^+ content of less than 1 mEq/L. Experimental rats (low Na^+ diet) were monitored for water consumption and weight gain at least 3 times per week until they attained a predetermined running weight (n = 16, average weight at run = 234.9 ± 7.6 g). To achieve this weight required an average of slightly more than 57 days. A second group of rats were placed on a semi-purified diet (US Biochem. Corp., Catalog Number 10662) which, by our analysis, delivered approximately 36 mEq/kg Na^+. These rats achieved their running weight in only 13 days. The average running weight of the animals in the low-sodium diet was 235 ± 7.6 g; for the control group this value was 236 ± 6.9 g, $p > 0.05$. Experimental and control rats were treated identically save for the dietary regimen.

On the day prior to an exercise bout rats were fitted with a permanent, indwelling catheter (Silastic, external jugular vein) for rapid and convenient blood sampling. Catheters were implanted aseptically under pentobarbital anesthesia. On the day of the experimental run each rat was equipped with a rectal probe (model # 701, Yellow Springs Instr. Co., Yellow Springs, OH) inserted to depth of 6 cm, and a surface probe (Yellow Springs, # 709) secured

mid-length on the tail. With the animal in a restraining cage, a small sample (0.8 ml) of blood was removed to determine pre-run or control levels of several clinical chemical indices of heat/exercise injury. Following this procedure, rats were rapidly removed to a large (3 m X 4 m) stainless steel chamber maintained at 35 $\pm$ 0.5°C, 25% RH. By a shock avoidance contingency rats were then run (9.14 m • min^{-1}, level treadmill, 35°C ambient) until hyperthermic exhaustion (T_{re} = 43°C) ensued. Rectal (T_{re}) and tail-skin (T_{sk}) temperatures were monitored on a minute-by-minute basis while the animals were exercising in the heat. Immediately upon completion of the run a post-run blood sample was removed; both blood samples were centrifuged (10000 g, 4°C), and the plasma was separated and frozen (-20°C) for subsequent analysis.

All plasma samples were analyzed for the commonly reported indices of heat/exercise injury (15, 17) as well as circulating levels of aldosterone and cortisol. Worthington test kits were used to quantitate circulating levels of glucose, lactic acid dehydrogenase (LDH), urea nitrogen, and creatinine, by methods outlined in the respective technical bulletins. Potassium (K^+) and sodium (Na^+) levels were analyzed by standard flame photometric techniques (Radiometer, Copenhagen) while lactate levels were estimated by means of test kits prepared by the Sigma Chem. Co. (St. Louis, MO). Plasma levels of cortisol were assayed by radioimmunoassay test kits manufactured by Damon Diagnostics (Needham, MA) while aldosterone concentrations were quantitated using radioimmunoassay test kits manufactured by Sorin Biomedica (Saluggia, Italy) and distributed by Damon Diagnostics. Both radioimmunoassay tests were carried out according to procedures described in the respective technical bulletins.

Statistical analyses were performed by analysis of variance (23) followed by Tukey's t-test to permit all pair comparisons (22); Student's non-paired t test was also used where appropriate. The null hypothesis was rejected at $p < .05$.

Results

Fig. 1 demonstrates that despite a large decrement ($p < 0.01$) in the rate of weight gained among the sodium deprived rats, both groups of animals consumed water at equivalent rates. It should be recalled, however, that at the time of the experimental runs, both groups of rats were at the same average weight. Fig. 2 demonstrates that, despite the sodium deprivation, experimental animals were able to perform on the treadmill for the same time interval as controls (p = ns) with an equivalent weight (water) loss during this interval, despite significant ($p < .001$) increments in hematocrit ratios (Fig. 3) among rats fed the low sodium diet. Additionally, this increased hematocrit in the low sodium group may have contributed to the significantly ($p < .001$) decreased final (maximal) skin temperatures achieved in this group (Fig. 4).

Table 1 demonstrates the effects of prolonged consumption of a diet deficient in sodium and exercise in the heat to hyperthermic exhaustion on several clinical chemical indices of heat/exercise injury and hormonal levels. Despite the prolonged interval on the low-sodium diets, the experimental animals successfully conserved Na^+; in both pre- and post-run blood samples there occurred no significant differences (p = ns) in Na^+ levels between the two groups. However, a significant ($p < .01$) increment occurred in both groups when pre-run levels were compared with the respective post-run values. This is also true of plasma K^+ levels in both groups of rats. Of considerable interest are the data which indicate (in the pre-run samples) that circulating levels of aldosterone were increased by 160% ($p < .01$) over control values in the low-sodium group. In both groups exercise in the heat to hyperthermic exhaustion resulted in significant increments ($p < .01$, pre vs post) in aldosterone levels. This increased adrenocortical activity was similarly manifested in circulating cortisol/corticosterone levels. In the control samples (i.e. pre-run) rats fed the

low Na^+ diet had significantly ($p < .05$) increased cortisol/corticosterone (+ 126%) concentrations. These increments were maintained after exercise in the heat to hyperthermic exhaustion (i.e. post-control vs post-low Na^+, $p < .01$) although both groups had proportional increases.

Consumption of the low Na^+ diet had a notable effect on lactate accumulation in the plasma. Thus, in both the pre-run and post-run samples lactate levels were significantly ($p < .01$) elevated in rats consuming low Na^+. In both groups the lactate levels in the post-run samples were significantly ($p < .01$) greater than in the respective pre-run samples. It is interesting to note that while there were no significant intergroup differences in pre-run levels of either circulating glucose or LDH, there occurred a significant ($p < .01$) difference in the post-run glucose levels while LDH was increased consistently in both groups. As expected, creatinine levels were significantly ($p < .01$) increased after exercise in the heat in both groups; of more interest is the observation that in both blood samples, circulating creatinine concentrations were significantly ($p < .01$) increased in the rats consuming the low sodium diet. Alternatively, circulating levels of urea nitrogen were unaffected by the low sodium diet, but were significantly ($p < .01$) elevated following exercise in the heat.

Discussion

Prolonged consumption of a diet deficient in sodium elicited a variety of notable observations: (a) severely restricted weight gain despite equivalent water consumption; (b) significantly increased hematocrit with concomitant reduction in T_{sk} during exercise in the heat; (c) no differences in endurance capacity or circulating Na^+ levels; (d) significantly increased adrenocortical activity effecting efficient Na^+ conservation; (e) increases in circulating lactate and creatinine levels in sedentary animals and decreased glucose concentration

following exercise to hyperthermic exhaustion. It is probable that plasma levels of Na^+ were maintained, not only by aldosterone-induced, extremely efficient renal reabsorption (2), but also by a reduction in plasma volume secondary to reduced extracellular fluid osmolarity (4). Thus, there would necessarily occur a fluid shift toward intracellular compartments in the rats on the sodium-deficient diet; such an occurrence would also partially explain the significant hemoconcentration, probably due to increased cell volume, manifested in the increased hematocrit ratios both pre-run and post-run in rats consuming the low sodium diet. Thus, the significantly increased hematocrit ratios observed in the Na^+ deprived rats could have contributed to decreased cutaneous circulation and peripheral heat dissipation. This, in turn, would have effected the notable decrements in tail-skin temperature that occurred during exercise in the Na^+ deprived group.

While both heat exposure (8, 20) and exercise (12) have been demonstrated to increase the secretion of the fluid regulatory hormones, the magnitude of these responses was enhanced by a diet low in sodium (1) and attenuated by dietary salt excess (3). The results of the present experiments indicated that prolonged feeding of a diet deficient in sodium increased the circulating levels of both aldosterone and cortisol/corticosterone. In pre-exercise plasma samples, we observed a 160 % increment in aldosterone levels in the low sodium group. Interestingly, following exercise in the heat to hyperthermic exhaustion, aldosterone levels were increased by only approximately 23% in the low-sodium group, but by nearly 140% in the controls. This result may be suggestive that mineralocorticoid secretion was nearly maximally stimulated in the low-Na^+ group over the extended dietary interval. Alternatively, stimulation of adrenocortical activity as a result of exercise in the heat to hyperthermic exhaustion was more consistent between groups. Post-exercise levels of

cortisol/corticosterone were increased by approximately 2.2 fold in the control group and 2.7 fold in the low-sodium group. We concluded generally from these results that increased mineralocorticoid secretion in the Na^+ deprived group contributed greatly to the conservation and maintenance of circulating Na^+. Because there were no significant differences in endurance capacity between groups, we might speculate that tissue levels of Na^+ were also reasonably well maintained. We had originally anticipated decreased run times in the Na^+ deprived group as a result of exercise/salt-depletion muscle cramping; however, this hypothesis was not substantiated by the data of these experiments.

The significantly increased levels of creatinine and lactate in the pre-run blood samples of the rats consuming low Na^+ might be explained by either a redistribution of blood flow, specific metabolic effects, or increased renal reabsorption in this group. Since long-term Na^+ deprivation stimulates extremely efficient tubular reabsorption of Na^+ (2) concurrent with water reabsorption and decreased urine flow (24), similar effects on circulating creatinine and urea nitrogen levels might be expected. While both are increased significantly by exercise in the heat to hyperthermic exhaustion, creatinine levels are affected by the low Na^+ diet while urea nitrogen levels are unaffected. Similarly, plasma lactate levels are markedly elevated by consumption of the low Na^+ diet. Since the liver and kidney are the major sites of uptake and utilization of lactate (21), increments in pre-run levels of lactate in the low Na^+ group might also be attributed to an effect of reduced sodium ingestion on splanchnic blood flow. However, once again, circulating levels of glucose in the pre-run samples are not different between groups. These apparent effects of the Na^+ deficient diet may be indicative of specific metabolic effects, heretofore unreported. Additional experiments are planned to elucidate further the metabolic and physiologic consequences of prolonged consumption of a Na^+ deficient diet.

Figure Legend

Fig. 1. Effects of prolonged consumption of a sodium deficient diet on weight gain and water consumption in immature male rats. Mean levels $\pm$ standard errors of the mean are depicted for an n of 16 in both groups.

Fig. 2. Effects of a sodium deficient diet on endurance capacity and weight loss during exercise in the heat. Rats were run at 9.14 m/min on a level treadmill at an ambient temperature of 35°C until hyperthermic exhaustion (T_{re} ~ 43°C). Remaining conditions are as noted under Fig. 1.

Fig. 3. Effects of a sodium deficient diet on hematocrit ratios immediately prior to and following exercise in the heat to hyperthermic exhaustion; all conditions are as noted under Figs. 1 and 2.

Fig. 4. Effects of a sodium deficient diet on final rectal and skin temperatures following exercise in the heat to hyperthermic exhaustion. All conditions are as noted under Figs. 1 and 2.

The authors gratefully acknowledge the skilled technical assistance of Natalie Leva, Virginia Pease, and Shawn Wright. We express our gratitude to Lianne Gallerani, Sandra Beach and Julie Cyphers for their technical assistance in preparing the manuscript. The views, opinions, and findings contained in this report are those of the authors and should not be construed as an official department of the Army position, policy, or decision, unless so designated by other official documentation.

In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals," as prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council.

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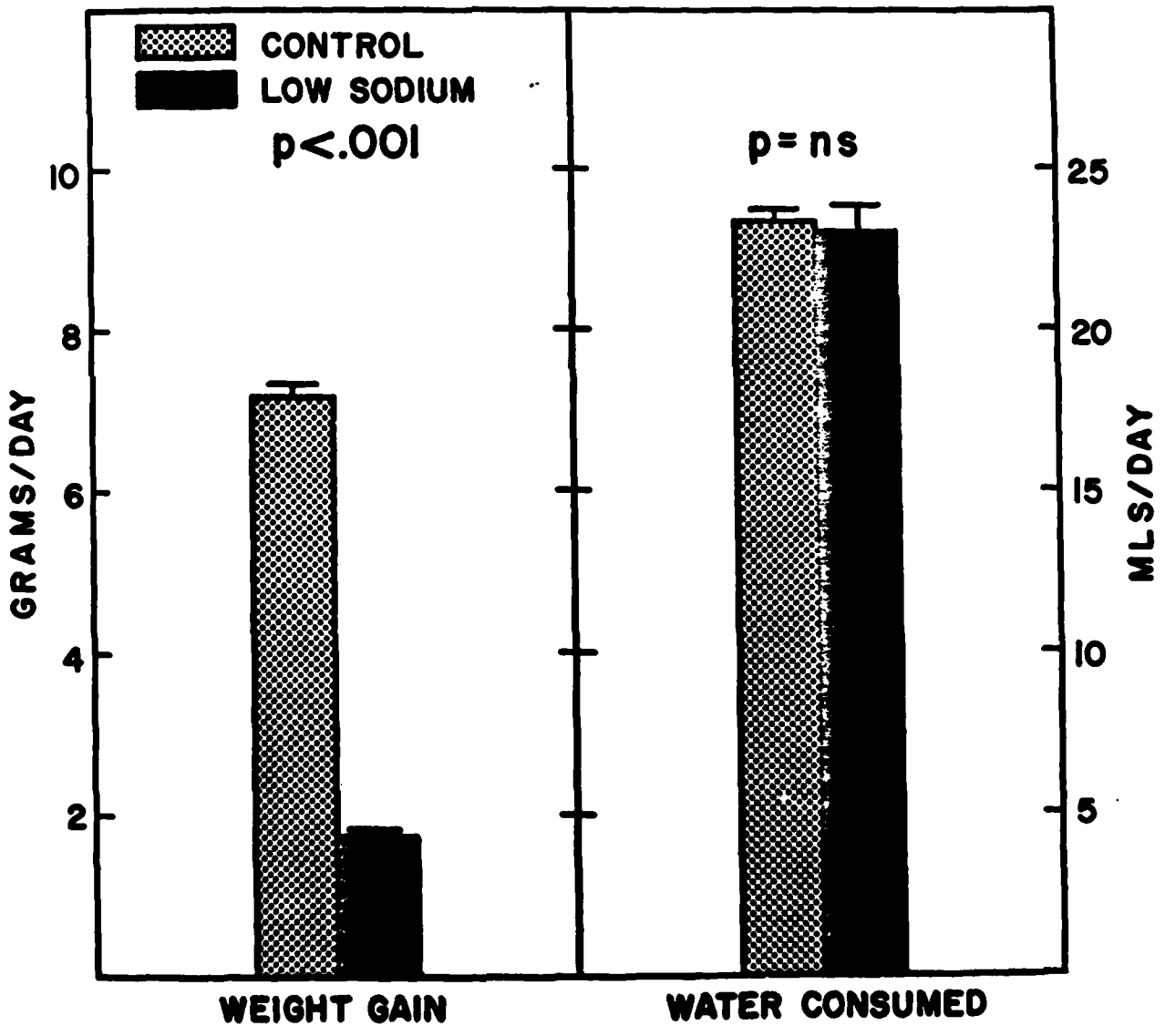
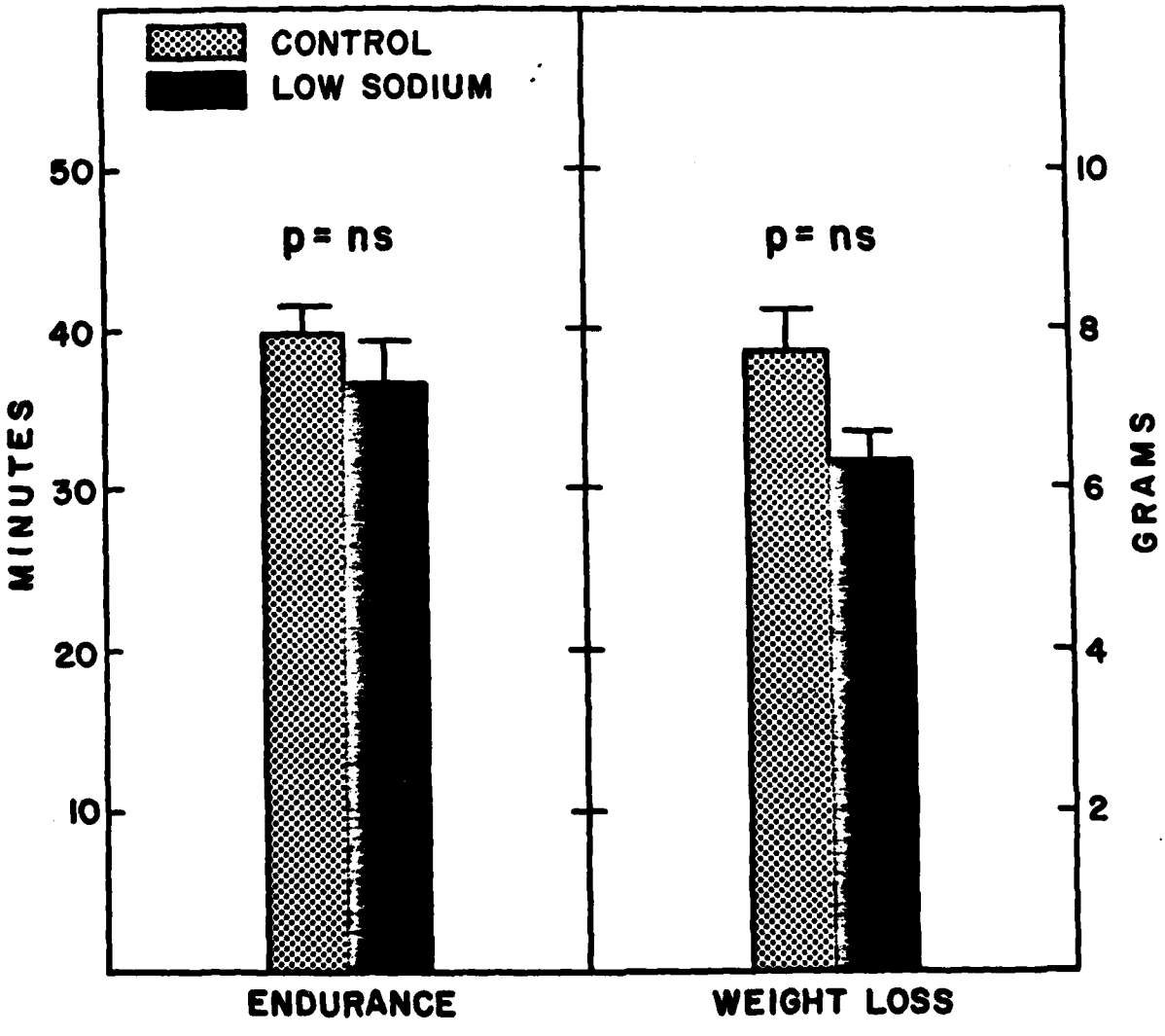
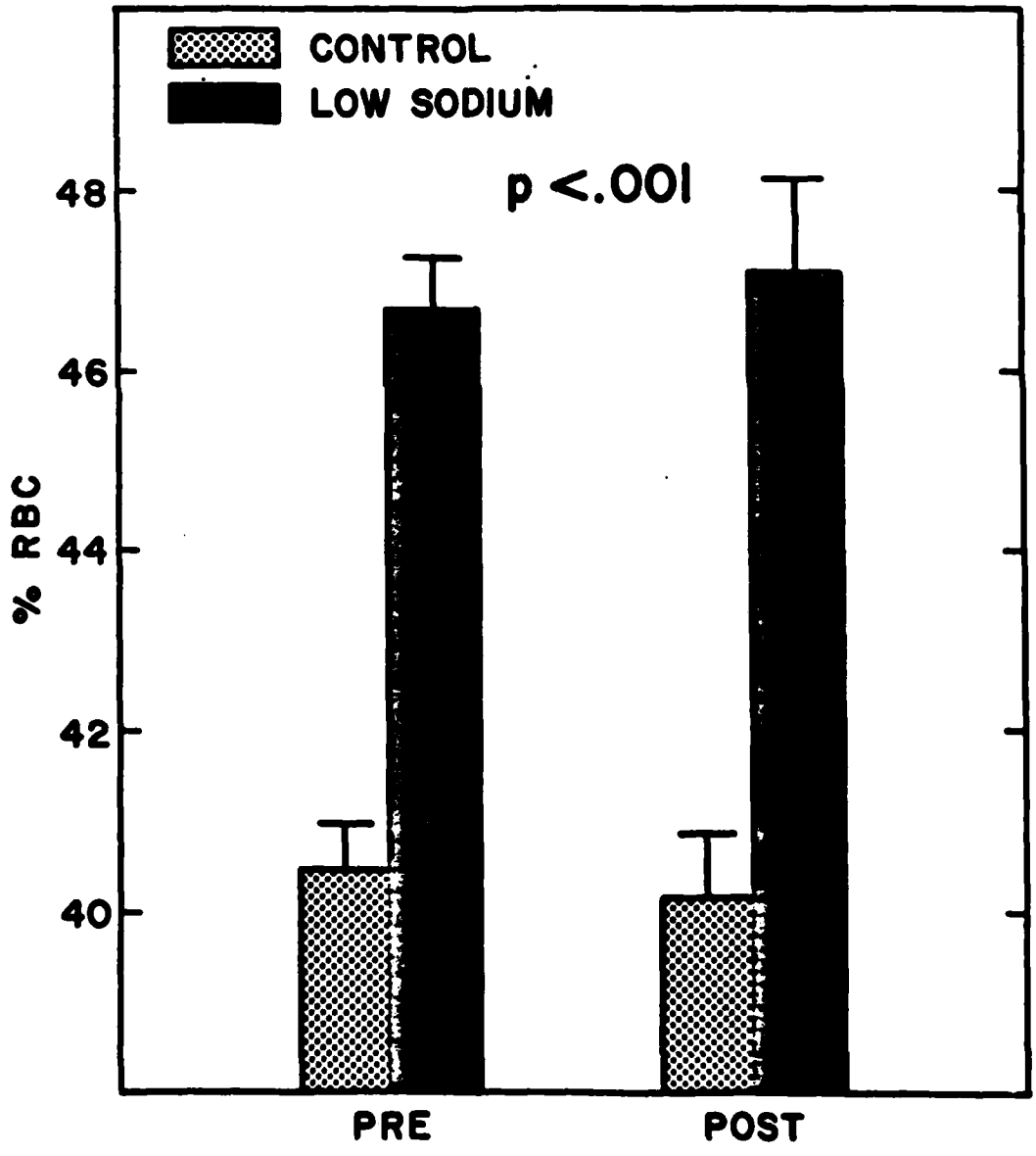
EFFECTS OF A LOW SODIUM DIET ON AVERAGE WEIGHT GAIN AND WATER CONSUMPTION



EFFECTS OF A LOW SODIUM DIET ON ENDURANCE AND WEIGHT LOSS DURING EXERCISE IN THE HEAT



EFFECTS OF A LOW SODIUM DIET ON HEMATOCRIT RATIOS PRIOR TO AND FOLLOWING EXERCISE IN THE HEAT



EFFECTS OF A LOW SODIUM DIET ON FINAL CORE AND SKIN TEMPERATURES FOLLOWING EXERCISE IN THE HEAT

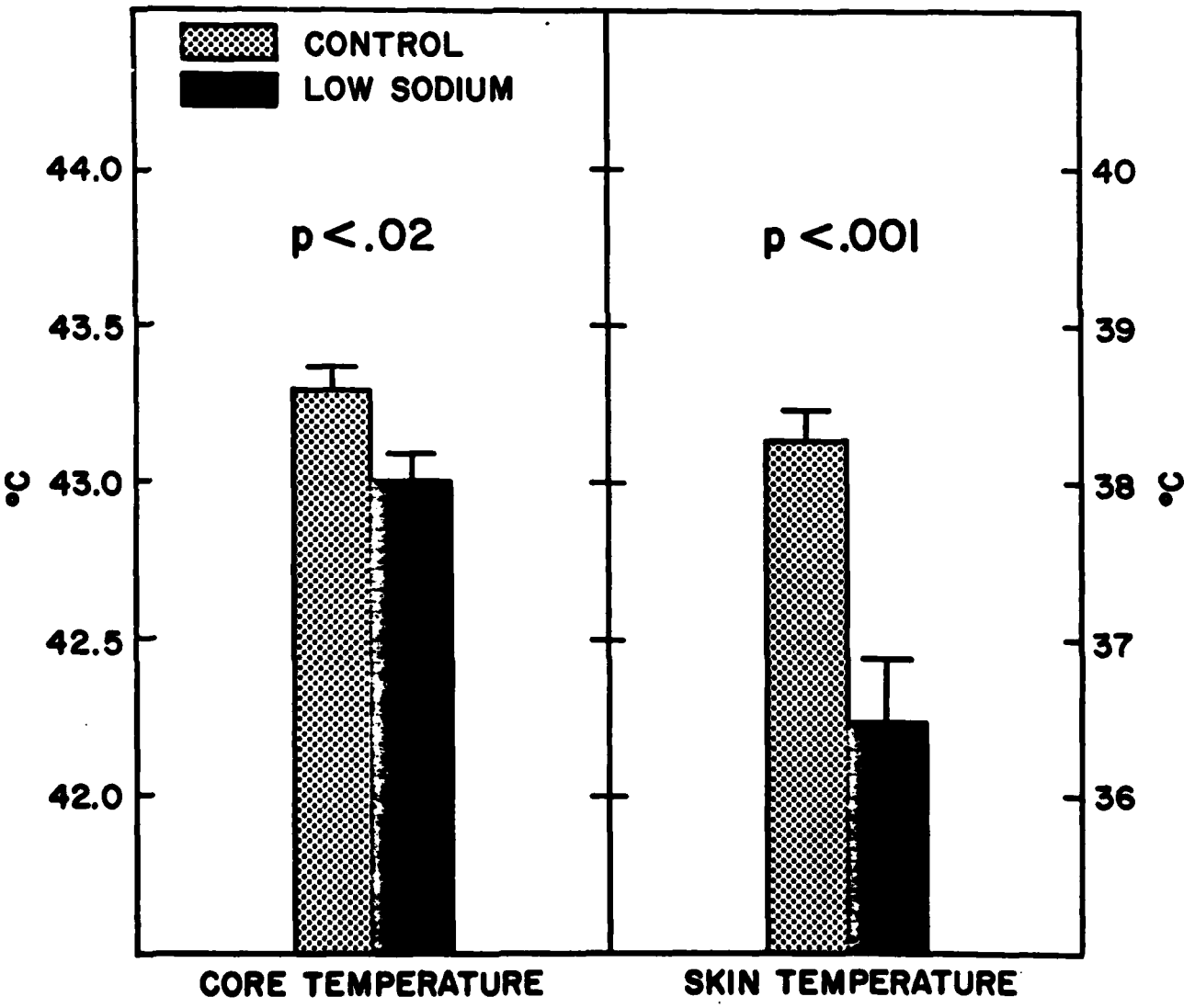


TABLE 1. EFFECTS OF A LOW SODIUM DIET AND EXERCISE IN THE HEAT TO HYPERTHERMIC EXHAUSTION ON CLINICAL CHEMICAL INDICES OF HEAT / EXERCISE INJURY AND ADRENOCORTICAL HORMONES IN PLASMA. BLOOD SAMPLES WERE TAKEN IMMEDIATELY PRIOR (PRE) AND SUBSEQUENT (POST) TO EXERCISE IN THE HEAT. MEAN VALUES ± SEM ARE NOTED FOR AN N OF 16 IN EACH GROUP, EXCEPT N= 6 (LDH) AND N=11 (CREATININE).

	CONTROL		LOW SODIUM	
	PRE	POST	PRE	POST
SODIUM (mEq/L)	142.5±.38	145.5±.38	140.69±.71	144.75±.79
POTASSIUM (mEq/L)	4.43±.10	5.47±.15	4.71±.13	5.88±.32
ALDOSTERONE (ng/ ml)	.38±.04	.90±.03	1.37±.06	1.69±.06
CORTISOL (μg/100ml)	1.65±.3	5.24±.51	3.73±.68	13.85±.7
LACTATE (mg/100ml)	27.4±1.8	52.9±5.2	65.9±7.5	101.1±7.4
GLUCOSE (mg/100ml)	145.4±3.3	176.3±15.2	153.1±7.3	123.1±10.1
LACTATE DEHYDROGENASE (IU/L)	96.0±17.4	532.4±111.3	82.6±20	557±155
UREA NITROGEN (mg/100ml)	13.6±.67	21.7±.97	11.7±.53	18.5±.8
CREATININE (mg/100ml)	.73±.03	1.31±.04	1.05±.05	1.67±.08